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INFANT MORTALITY IN RURAL BANGLADESH: STATE DEPENDENCE VS. UNOBSERVED HETEROGENEITY

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Infant mortality in Rural Bangladesh: State Dependence vs. Unobserved Heterogeneity[a]

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Abstract

Using longitudinal data of the Health and Demographic Surveillance System (HDSS) in Matlab, Bangladesh, covering the time period 1982 – 2005, and exploiting dynamic panel data models, we analyze siblings' death at infancy, controlling for unobserved heterogeneity and a causal effect of death of one child on survival chances of the next child. Matlab is a rural area split into two: a "treatment" area where along with standard government services extensive maternal and child health interventions are available, and a "comparison" area where only the standard government services are available. The observed infant mortality rates are 50 per 1,000 live births in the treatment area and 67.4/1,000 in the comparison area. We use separate models for the two areas and analyze the differences in infant mortality between the two areas using several decompositions.

Our model predicts that in the comparison area, the likelihood of infant death is about 30% larger if the previous sibling died at infancy than if it did not, and the estimates suggest that, in the absence of this "scarring" effect, the infant mortality rate among the second and higher order births would fall by 6.2%. There is no evidence of such a scarring effect in the treatment area, perhaps because learning effects play a larger role with the available extensive health interventions. We find that distance to the nearest health clinic can explain a substantial part of the gap in infant mortality between the two areas.

Key words: childhood mortality, millennium goals, death clustering, dynamic panel data models

JEL codes: I12, J13, C33

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1. Introduction

By 1993-1994, according to the first Demographic and Health Survey (DHS) in Bangladesh, the child mortality rate (mortality before reaching the age of five years) was 133 per one thousand live births and the infant mortality rate (mortality before reaching the age of one year) was 87 per one thousand live births (reference period 1989-1993). Infant deaths thereby accounted for 65 percent of all under-five deaths. Since then Bangladesh recorded a sharp decline in under-five deaths – with 65 deaths per one thousand live births in the period 2002-2006 and a 0.5 percentage points decline each year. In contrast, infant deaths declined by only 0.2 percentage points (from 87 to 52 deaths per one thousand live births) over the same period. In view of the millennium development goal to reduce under-five mortality in Bangladesh by two thirds between 1990 and 2015 (see United Nations, 2001) further reduction of these rates remains an important target.

There is a considerable amount of research on the determinants of infant mortality, focusing on, for example, the fact that mothers who get their children at a younger age and mothers with little or no schooling are at higher risk. Recent demographic data from a wide range of countries have revealed that child deaths are clustered within families, due to observed and unobserved characteristics of the mother, the family, or the local community, and possibly also due to a causal effect of death of one child on the survival chances of later siblings (Das Gupta, 1990; Guo, 1993; Zenger, 1993; Sastry, 1997; Arulampalam and Bhalotra, 2006; Omariba et al., 2008). Following the recent work on siblings' death-clustering by Arulampalam and Bhalotra (2006), which is a methodological improvement over the previous studies, our paper investigates interfamily heterogeneity in infant deaths in a rural area in Bangladesh. The main methodological differences with Arulampalam and Bhalotra (2006) are twofold. First, prospective data are used rather than retrospective data on birth histories, making it possible to use explanatory variables that vary over time and are measured around the time of each child birth rather than only at the time of a retrospective survey. Second, the data cover two different areas, one with standard government health services and one with more extensive surveillance and health care, allowing for an analysis of the differences between the areas and giving insight in the consequences of providing extra health services.

The data come from the Health and Demographic Surveillance System (HDSS) in Matlab, a rural area in Bangladesh, with regular collection of prospective data for birth, death and other relevant information. HDSS is split up into two areas: the so-called ICDDR,B area

(the treatment area) and a comparison area. A general decline in under-five mortality took occurred in both areas until 1990, except in 1984 when the Shigella epidemic peaked. Since then an impressive decline in under five mortality is observed, resulting in a mortality rate of 45.3 per one thousand live births in the ICDDR,B area and 60.2 per one thousand live births in the comparison area in 2005 (HDSS, 2006).

Our main findings are the following. After controlling for all observed and unobserved differences between mothers, there is evidence of "scarring" - a negative causal effect of infant death on the survival chances of the next sibling - in the comparison area. No evidence of scarring is observed in the ICDDR,B area, where health care facilities are better; perhaps this is because learning effects play a role with the available extensive health interventions. Conditional on other covariates, we find that boys are more likely to die in the ICDDR,B area whereas no gender differences are found in the comparison area. The probability of infant death falls with the education level of the mother, particularly in the ICDDR,B area. Mother specific unobserved heterogeneity is found to play a significant role – it captures about 18 percent of the total unsystematic variation in the ICDDR,B area and 8 percent in the comparison area. Decomposing the gap in infant mortality between the two areas into several shows that distance to the nearest health clinic can explain a substantial part of the gap.

The remainder of our paper is organized as follows. Section 2 briefly discusses the related literature. Section 3 presents the data source. The empirical model is explained in Section 4. Section 5 introduces the variables used in the empirical model and presents some descriptive statistics. Estimation results are discussed in Section 6. In Section 7, decompositions of the mortality differential between treatment and comparison area are performed. Section 8 interprets some of our main findings and concludes.

2. Background

Death-clustering of siblings is widely noticed in the demographic literature of many developing countries, including Bangladesh (Hobcraft et al., 1985; Koenig et al., 1990; Das Gupta, 1990; Sastry, 1997; Guo and Rodriguez, 1992; Miller et al., 1992; Curtis et al., 1993; Zenger, 1993; Guo, 1993; Majumder et al., 1997; Alam and David, 1998; Arulampalam and Bhalotra, 2006, 2008; Bhalotra and van Soest, 2008; Omariba et al., 2008). Possible factors explaining death clustering are that siblings share the same genetic traits; that the mother has similar problems at several births such as premature delivery or

intrauterine growth retardation; maternal inability to take care of the child or manage the household;[d] and environmental factors such as poor water supply.

Death clustering of siblings can also be due to a causal process called state dependence. Arulampalam and Bhalotra (2006) refer to the notion that the death of one child may result in a higher risk of death for the next child as *(positive) scarring*. An explanation of state dependence is that a child's death leaves the mother depressed as a result of which her subsequent child's health is compromised in both womb and infancy (Steer et al., 1992; Rahman et al., 2004). This is referred to as the *depression hypothesis*. Another explanation of positive scarring is that women whose child dies have their next birth sooner (the *replacement hypothesis*), and the resulting closely-spaced pregnancies may lead to nutritional depletion which affects the health of the next born child (Gyimah and Rajulton, 2004; Hobcraft et al., 1983; Cleland and Sathar, 1984; Koenig et al., 1990; Zenger, 1993; Miller et al., 1992; Da Vanzo and Pebley, 1993).

Alternatively, one might also expect "*negative scarring"* mechanisms, in the case of competition for the use of family resources: if the previous child has died, the next child competes with fewer siblings, potentially improving its survival chances. Learning effects may also lead to *negative scarring*. For example, if the older sibling dies because of diarrhoea or acute respiratory illness (ARI), the mother may then learn how to prevent that her next child dies from the same cause.

Demographers using data on siblings' death-clustering have long been interested in knowing whether unobserved factors at the family level, such as genetic factors, lead to biased parameter estimates (estimates without accounting for the correlation among deaths of siblings), and spurious correlation (reverse causality), which may have important implications for conclusions concerning policy design. The conventional statistical tools in previous studies on child mortality (e.g., DaVanzo et al., 1983; Hobcraft et al., 1985; and Koenig et al., 1990) often made the assumption that unobservables in the death risk of consecutive children are independent of each other, and this may lead to biased estimates if mother specific unobserved heterogeneity plays a role (Guo, 1993).

[d] For example, Das Gupta (1990) argues that some women are less resourceful in caring for their children and managing household activities.

Zenger (1993) discussed different statistical methods for accommodating the correlation structure of the death of siblings. The first approach is to estimate a marginal logistic model, which avoids the problem of misspecification because no assumptions are made about the form of correlation. The second approach is to accommodate the correlation by including the survival outcomes of older siblings as explanatory variables in the regression model, leading to a transition model or Markov model. The third approach is known as the random intercept model, which allows for unobserved family specific mortality risks that follow some probability distribution. However, the models Zenger estimated included either the previous child's survival status or unobserved heterogeneity but, in no case, both.

Guo (1993) and several other studies (Curtis et al., 1993; Sastry, 1997; Bolstad and Manda 2001) have included survival status of the preceding sibling in the model allowing for unobserved heterogeneity. They did not, however, interpret these effects in terms of causality and correlation. According to Arulampalam and Bhalotra (2006), the estimated coefficient on the survival status of the previous sibling was biased in all these studies. Additional studies on siblings' death-clustering, including some based upon data from Matlab, discarded the first-born child in the family, implying that the estimates suffer from an initial conditions problem (Heckman, 1981), resulting in a potential bias in the estimates.

As an important methodological development, Arulampalam and Bhalotra (2006) developed a new way of consistently estimating models incorporating both previous sibling survival status (as a lagged dependent variable) and unobserved heterogeneity and, in addition, interpreted the former in terms of a causal process. They addressed the issue of initial conditions by modelling the birth of the first child and rejected the null hypothesis of an exogenous initial condition (no correlation between the family level unobservable and the survival status of the first child), implying that studies not accounting for the initial condition indeed lead to biased estimates. Their modelling approach forms the basis of our paper. It was used earlier by Arulampalam and Bhalotra (2008) and Omariba et al. (2008) and extended to incorporate birth spacing and fertility by Bhalotra and van Soest (2008).

3. Data

Health and Demographic Surveillance System, Matlab

Since 1966, the International Centre for Diarrhoeal Disease Research, Bangladesh (ICDDR,B) has maintained a Health and Demographic Surveillance System (HDSS) in Matlab, a typical rural area located 60 km southeast of Dhaka in which all births, deaths, causes of deaths (through verbal autopsy), pregnancy histories, migrations in and out of the area, marriages, divorces, and several indicators of socioeconomic status are recorded for the complete population of about 220,000 people.

ICDDR,B started the Maternal Child Health and Family Planning Programme (MCH-FP) project in October 1977 in half of the HDSS area, formerly known as MCH-FP area and currently as ICDDR,B area, which enhanced government health services and collected additional data on a range of health indicators – immunization status with specific date, breast-feeding, morbidity status (e.g., Diarrhoea, ARI), causes of death (based on verbal autopsy), and MUAC measures for nutrition status. The other half of the area, known as comparison area, remained under the usual programme of the Government of Bangladesh. Health and Demographic data have been collected systematically through regular household visits (every 2 weeks until January 1998, and once every month since then).

At each birth, the child is registered and the mother is asked about her previous pregnancy histories, including live births, gender, deaths, and stillbirths. Furthermore, causes of death are matched with the mother's pregnancy history. Pregnancy history variables provide us with all information on the children of a woman if all the births the woman gave took place in the HDSS area and were registered at birth. Alternatively, if a woman migrated out and gave birth outside of the HDSS area and again migrated in with the child at age below five years, the child was still registered (birth date, survival status, etc.) in HDSS. Otherwise, the child's records are not registered in HDSS, leading to incomplete records for mothers who did not always live in the HDSS area.

Study Sample

We combined the health and demographic surveillance system data from 70 villages in the ICDDR,B area and 79 villages in the comparison area obtained from 1 July, 1982 until 31 December, 2005 (the study period). Data from before 1 July 1982 have not been (yet) made available for research.

The complete data set has records on about 63,000 mothers, with more than 165,000 child births – including live singleton births, multiple births, and still births. For our purposes, however, we selected a subsample of mothers without multiple births[e] and with complete[f] live birth information who were continuously living in the Matlab area after the birth of their first child. This implies that we deleted mothers who migrated out of Matlab during the period under study. Moreover, we discarded stillbirths.[g] Finally, we have excluded the children of three villages which shifted from the ICDDR,B area to the comparison area in 2000. This leads to working samples of 31,968 children and 13,232 mothers in the ICDDR,B area and 32,366 children and 11,856 mothers in the comparison area, with the mothers in both areas residing continuously in the same area during the whole study period and having all their births in the same area.

4. Model Specification

This paper models the propensity of death in infants among Bangladeshi families, allowing for the identification of state dependence (scarring) and taking account of the potentially confounding effects of unobserved inter-family heterogeneity. State dependence refers to whether the survival status of the previous child (t-1) of a family (i) has an influence on the death of the next child (t) at infancy.

Let there be T_i children born alive in family i (i=1, 2,…,N – the number of families or mothers in the sample). Let t=1, 2,…,T_i denote birth order. The unobserved propensity to experience an infant death, y^*_{it}, is specified for children of birth order 2 or higher as

$$y^*_{it} = x'_{it}\beta + \gamma y_{it-1} + \alpha_i + u_{it} \quad \text{.....(1)}$$

[e] We eliminated multiple births as children of a multiple birth face much higher odds of dying requiring a separate analysis, as has been documented in the demographic literature.

[f] To have complete birth information of a woman during the study period we have calculated parity (total number of live births) from the pregnancy history variables. We have checked parity and birth dates of all children. For example, if a mother has parity four this means this mother has had four live births, so the birth dates of four children should be available in the file and this mother will appear four times as giving birth. If this was not the case (e.g., if a child was born outside of the Matlab area or before study period or deleting multiple births may caused incomplete birth information of a mother), we have deleted all children's records of this mother.

[g] One reason why we eliminated stillbirths from the data is that gender is an important covariate in our analysis but gender is missing for stillbirths.

Here y^*_{it} is the unobserved propensity of infant death. The observed infant death outcome $y_{it} = 1$ if the child's propensity for death crosses a threshold normalized to zero, that is, if $y^*_{it} > 0$; otherwise, if $y^*_{it} \leq 0$, $y_{it} = 0$ and the child does not die in infancy. x_{it} is a vector of strictly exogenous observed explanatory variables and β is the vector of coefficients associated with x_{it}. The term α_i captures unobserved heterogeneity at the family (mother) level which remains the same for all births of a given mother, accounting for all unobservable family characteristics including genetic characteristics and variables such as innate maternal ability which influence the index child's propensity to die. The coefficient γ is associated with state dependence – the effect of death in infancy of the previous child on the next child's survival chances - and the null hypothesis of no state dependence implies that $\gamma=0$.[h]

The model assumes that the history of infant deaths among older children other than the immediately preceding child has no direct effect on $y^*_{it.}$ For example, if child t-2 died in infancy then in our model this will affect the risk of death of child t-1 and, thereby, also the risk of death of child t, but there is no *direct* effect on death of child t. This is the first order Markov assumption (Zenger, 1993; Arulampalam and Bhalotra, 2006).

The model can be seen as a dynamic binary choice (unbalanced) panel data model, where the cross-section units are mothers (i) and birth order replaces time (t). Such models have been studied and applied in numerous studies (Hsiao, 1986, and Wooldridge, 2002), e.g. in the context of unemployment scarring (Heckman, 1981, and Stewart, 2007), and have recently also been used to analyze clustering of infant deaths in India and Kenya using retrospective data on birth histories in cross-sections of the Demographic and Health Survey (Arulampalam and Bhalotra, 2006, 2008; Omariba et al., 2008). The current paper extends the proposed model in terms of the covariates used, exploiting the data from HDSS which are collected prospectively. All time-varying covariates in the model except access to piped water are collected at each time the mother gave birth. This data have an advantage compared to retrospective survey data in terms of time consistency of dependent and independent variables, enriching the set of covariates that can be used without the introduction of measurement error.

[h] In principle it would be possible that child t dies in infancy before child t-1 does, violating the sequence of events assumed in our model. This never happens in our data and is therefore ignored.

With the above specifications the conditional probability of death for an infant t of mother i, given $y_{it-1,}$ x_{it}, and α_i x_{it}, is given by:

$$P[y_{it}=1| y_{it-1,} x_{it}, \alpha_i] = \Phi [(x'_{it}\beta + \gamma y_{it-1} + \alpha_i)] \ldots\ldots\ldots\ldots\ldots\ldots\ldots\ldots\ldots\ldots\ldots\ldots\ldots\ldots(2)$$

where Φ denotes the cumulative distribution function of the standard normal distribution.

The joint conditional probability of the observed sequence of binary outcomes given α is given by:

$$P(y_{i1,\ldots\ldots\ldots,} y_{i,T(i)} | \alpha_i, x_{i1}, \ldots, x_{iT(i)}) =$$
$$P(y_{iT(i)}|y_{iT(i)-1,} \alpha_i, x_{iT(i)}) P(y_{i,T(i)-1}|y_{i,T(i)-2,} \alpha_i, x_{i,T(i)-1})$$
$$\ldots\ldots P(y_{i2}|y_{i1,} \alpha_i, x_{i2}) P(y_{i1}| \alpha_i, x_{i1}) \ldots\ldots\ldots\ldots\ldots\ldots\ldots\ldots\ldots\ldots\ldots\ldots\ldots\ldots(3)$$

It is clear from the sequence above that it is necessary to give a specification for $P(y_{i1}|\alpha_i,x_{i1})$ (the "initial condition problem" in dynamic models with unobserved heterogeneity (e.g. Heckman, 1981). Modelling the outcome for the first child is especially relevant because the first child shares unobservable traits α_i with its younger siblings. If there were no unobserved heterogeneity (α_i,=0 for all i) then the initial observation could be treated as exogenous, and the model that is given in equation (1) could be estimated by using the sample of second and further children. Alternatively, Hsiao (1986) showed that the initial condition problem can be ignored even with unobservable heterogeneity if the time dimension of panel (T(i)) is large, but in our case T(i) is the total number of children born in family i, and this is typically small, so that asymptotic results based upon large T will not apply. Since the correlation between α_i and y_{it-1} that makes y_{it-1} endogenous in equation (1) is probably positive, ignoring it would probably lead to overestimation of the state dependence coefficient γ (Fatouhi, 2005). This is why we specify a separate equation for the risk of mortality of the first-born child of each mother. The equation for the process of generating first observations will have the same form as for equation (1)[i] and is given by

[i] See Arulampalam (2007) for a discussion of alternative approaches to modeling the initial condition.

$$y^*_{i1} = x'_{i1}\pi + \theta\alpha_i + u_{i1} \quad \text{...(4)}$$

Exogeneity of first child survival corresponds to $\theta=0$ which can be tested in a standard way. The distribution function is same as for equation (1). The probability of infant death for the first born child corresponding to equation (4) is given by:

$$P(y_{i1}=1|\alpha_i, x_{i1}) = \Phi\, [x'_{i1}\pi + \theta\alpha_i] \quad \text{..(5)}$$

Assuming the error terms u_{i1} and u_{it} for t>2,…., T(i) are independently distributed following standard normal distributions, combining the equations (1) and (4) gives a complete dynamic model for all observations with observed and unobserved heterogeneity at family level i. The conditional probability of an observed sequence of binary outcomes $y_{i1,\ldots,}\ y_{iT(i)}$ for infant survival and deaths for all children of family i can be written as:

$$P\,(y_{i1,}y_{i2,\ldots}y_{iT(i)}\,|x_{iT(i)},\ldots,\,,x_{i1,}\,\alpha_i) =$$
$$\Phi\{(x'_{i1}\pi + \theta\alpha_i)(2y_{i1} - 1)\}\Pi\{\Phi(x'_{it}\beta + \gamma y_{it-1} + \alpha_i)(2y_{it} - 1)\} \quad \text{..............(6)}$$

and marginalizing the likelihood with respect to the unobserved heterogeneity component α_i gives the following likelihood contribution for family i:

$$L_i = \int P\,(y_{i1,}y_{i2,\ldots}y_{iT(i)}|x_{iT(i)},\ldots,x_{i1,}\alpha) f(\alpha)\, d\alpha \quad \text{..(7)}$$

where $f(\alpha)$ is the probability density function of α, which is taken to be normal with mean 0 and variance σ^2_α independently identically distributed and independent of all other observables and unobservables. The integral in (7) is computed using Gauss-Hermite quadrature (Butler and Moffitt, 1982).

The joint random-effects dynamic probit model taking account of initial conditions is non-standard and cannot be estimated using the routines available in standard statistical software. Stewart (2007) has written Stata code for fitting the random-effects dynamic probit model, and we have fitted this model in our data. Our results are based on specifying 32 quadrature points.

5. Variables and Descriptive Statistics

The dependent variable infant death (y_{it}) is defined as 1 if the child is observed to die before the age of 12 months and as 0 otherwise. One of our main interests is in the effect of the lagged dependent variable $y_{it-1,}$ the infant survival status of the preceding sibling. The other explanatory variables are included in x_{it}.

All child specific covariates x_{it} are measured at the time of birth: birth order of the child, gender, and the age of mother at birth of the index child; education of the mother is denoted by a set of dummy variables for the years of schooling attained (no schooling (the omitted category), 1 to 5 years of schooling, or 6 or more years of schooling). The mother's education level may proxy her ability to take good care of her children but may also proxy the family's socio-economic status. Similarly, education and occupation of the father are included with a set of dummy variables, mainly reflecting the family's socioeconomic status.

Following Arulampalam and Bhalotra (2006), birth intervals are not included. Our estimates of the effect of scarring will therefore include the potential effect through replacement – if infant death reduces the time until the next conception due to a desire to replace the child that was lost, and a short birth interval increases the probability of infant death, then this is one mechanism that leads to positive "scarring". On the other hand, it also makes the birth interval endogenous (e.g., it is correlated with the mother specific effect in the infant mortality equation) so that explicitly separating this effect from other scarring effects requires a more complicated model (cf. Bhalotra and van Soest, 2008).

The mother's birth cohort also enters the model, giving insight in the trend of scarring over time. Another family level covariate is religion: following Bhalotra et al. (2008) who find that in India, Muslims have lower mortality probabilities than otherwise similar Hindus, we include a dummy for Muslims. More than 80% of the mothers in our sample are Muslims, the others are mainly Hindus.

To control for environmental factors, we include a dummy for access to running drinking water (piped drinking water / tube well). Moreover, we include the distance to the nearest health facility, defined as a sub-centre or ICDDR,B hospital in the ICDDR,B area and a

Upazila Health Complex in the comparison area.[j] This variable differs substantially between the comparison area and the ICDDR,B area, because of the additional health facilities in the latter.

A profile of both areas is given in Table 1, presenting percentages of outcome 1 for dummy variables and sample means for the other variables. The average number of children born per mother is 2.42 in the ICDDR,B area and 2.73 in the comparison area; 19 percent of families had more than three children in the ICDDR,B area, compared to 29 percent in the comparison area. 82.7 percent of all women in the ICDDR,B area and 89.8 percent of the comparison sample are Muslims. No differences are observed in average schooling years or mothers' age at birth between the two samples. A somewhat higher percentage of women in the comparison area are categorized as "no schooling." This includes those who attended Maktab/Madrasa, academic institutions where religious education is given. Sources of drinking water use are categorized into two as 0 'pond/river/tank' versus 1 'tubewell/filter'. The comparison area mothers less often have access to the more hygienic source of drinking water (tubewell/filter). Mothers residing in the ICDDR,B area are much nearer to a health facility (2 kilometres on average) than their counterpart mothers in the comparison area (7 km on average).

In the ICDDR,B area sample, a total of 1,599 (5.00% of all births) infant deaths in the sample occurred to 1,390 families (10.50% of all families), so that 11,842 (89.50%) families had no experience of infant death. Moreover, 0.01 percent of all families lost all their children in infancy. The percent of first born children is 41.4 and the percent of infant death of first born is 6.63, which is substantially higher than the infant death rate of all children (5.00%).

In the comparison sample, 2,180 (6.74% of all births) infant deaths occurred to 1,834 families (15.47% of all families); the remaining 84.53 percent of all families did not experience any infant deaths. Like in the ICDDR,B area, 0.01 percent of all families lost their all children at infancy, and the percentage of infant death is higher among the first born children than among other children (8.78% for first born; 5.55% for other children).

[j] The health facilities offer emergency obstetric care (EOC), antenatal care, delivery, referral and contraceptive services, counseling on side effects of contraceptive use, and health education. In addition, children with minor illnesses are treated, while children with severe illnesses are diagnosed and referred to a hospital. Children suffering from malnutrition are also treated.

Among families experiencing infant deaths, about 13.2% had more than one death in the ICDDR,B area, compared to 26.2% in the comparison area (not shown in the table).

Figure 1 presents the infant mortality rates by year of (child) birth for the treatment and comparison area. It clearly shows a decreasing trend in both areas until the late nineties. The infant mortality rate has always been higher in the comparison area than in the treatment area. At least in absolute terms, the differential has fallen over time, but both the levels and the difference seem to have stabilized since the late nineties.

Table 2 shows the raw probabilities of infant death conditional on the survival status of the preceding sibling. Explaining this is one of the primary goals of this paper. The probability of infant death is higher by 4.42%-points (7.96% rather than 3.54%) if the preceding sibling died as an infant in the ICDDR,B area, and by 5.07%-points in the comparison area (10.17% rather than 5.10%). In other words, the likelihood of infant death is 2.25 times higher in the ICDDR,B area and 2.00 times higher in the comparison area if the preceding sibling died than if it survived.

6. Estimation Results

Several dynamic random effects probit models that incorporate the lagged dependent variable (survival status of the previous sibling) and unobserved heterogeneity are estimated. The first (Model 1) includes only the infant survival status of the previous sibling (y_{it-1}); the second (Model 2) adds both child-level and mother-level factors, and the third also adds father-level factors (Model 3).

The results are presented in Table 3a (equation for children of birth order larger than one) and Table 3b (equation for the first born child). The results of Model 1 with only the lagged dependent variable (with parameter γ) show that the death of the immediately preceding sibling had a positive and significant effect (p=0.001) on the conditional probability of infant death in the comparison area, whereas a positive but insignificant effect (p=0.401) is observed in the ICDDR,B area.

The partial effect of y_{it-1} on $P[y_{it}=1| y_{it-1}, x_{it}, \alpha_i]$ can be derived from the estimates by constructing counterfactual outcome probabilities $p_0, p_1,$ taking y_{it-1} as fixed at 0 and 1, and evaluated at the overall means of the exogenous variables ($x_{it}=x..$). The difference

between p_0 and p_1 can be interpreted as average partial effect (APE); the ratio of the two is the predicted probability ratio (PPR) (Stewart 2007, p.522). Both are indicators of state dependence or scarring. In Model 1, the APE is about 2.16% in the comparison area whereas it is less than 1% in the ICDDR,B area (see Table 3c). In terms of PPR, the state dependence effect implies that the likelihood of infant death is about 42% larger if the older sibling died at infancy in the comparison area and about 14% in the ICDDR,B area.

In the comparison area, including child and mother-level variables reduces the parameter estimate of γ and its significance level (p=0.04) (Model 2); adding the father's characteristics leads to a small increase of γ and its significance level (p=0.03). In the ICDDR,B area, adding the regressors in Models 2 and 3 leads to small negative and insignificant estimates of the effect of previous sibling's death. In fact, though the sign changes, all three models in the ICDDR,B area find an insignificant effect of previous sibling's death (Table 3a). The predicted probability ratios (PPR) in Table 3c show that according to model 3, the likelihood of infant death in the comparison area is 30% higher if the previous child died at infancy than if it was alive. This effect is smaller than the estimate of Model 1, due to including the covariates.

The second panel (b) of Table 2 is based upon the estimation results of Model 3. Comparing the estimated average partial effect (APE) reported in the second panel of Table 2 with the difference in the probabilities in the first panel that only condition on previous child survival status shows that in the comparison area, almost one third (30% - row 11) of infant death clustering is a scarring effect. The remaining part is explained by observed and unobserved heterogeneity. In the ICDDR,B area, the estimated scarring effect is -14% but not significant).

Comparing the predicted probability of infant death (excluding first-borns) with the predicted probability of infant death when the previous sibling was alive gives an estimate of the reduction in mortality that would be achievable if scarring were eliminated (γ=0). The estimates suggest that, in the absence of scarring, the infant mortality rate among children of birth order two and higher would fall by 6.24% in the comparison area (row 13, Table 2).

We can test whether the initial period outcome (survival of the first child) within a family can be treated as exogenous. If θ=0 in equation (4), then unobservables in the equation for

the first observation are uncorrelated with unobservables in the (dynamic) equation for subsequent observations, and in this case there would be no need for the specification of separate equation for the first observations (Stewart, 2007; Arulampalam and Bhalotra, 2006). The null hypothesis $\theta=0$ is firmly rejected for all our models in the ICDDR,B area as well as the comparison area; see Table 3b). This confirms the importance of accounting for the initial condition.

The proportion of the total unsystematic variance that is attributable to family-level unobservables α_i is estimated to be 8% in the comparison area and 22% in the ICDDR,B area. The estimates decisively reject the null hypothesis of no family-level unobservables in both areas (Table 3b). Accordingly, in pooled probit models that ignore family-level unobserved heterogeneity, the effect of previous sibling's death was overestimated: the estimate of γ for the comparison area was much higher there (0.2828) than in the complete model (0.1354; Model 3); in the ICDDR,B area, this difference was even larger (0.2996 versus –0.0918).[k] This shows the importance of controlling for α_i in the analysis.

The other covariates often play different roles in the treatment and comparison area and for children of first and higher birth orders. Among first born children, sons are more likely to die than daughters in both areas, but the difference in the comparison area is smaller than in the ICDDR,B area and only marginally significant. No significant gender differences are observed for higher birth orders. In the ICDDR,B area, the probability of infant death is U-shaped in the mother's age at the time of child birth, with a minimum at about 30 years of age. In the comparison are, the pattern is similar for first born children but there is no evidence of increasing death probabilities at older ages for children of higher birth orders. The mother's birth cohort dummies (where the reference category is the cohort of mothers born before 1966) consistently indicate significantly lower infant mortality probabilities for younger cohorts in both areas and for first born as well as higher birth orders, probably because of a time trend in hygienic circumstances and health technology.

[k] According to Stewart (2007) and Omariba et al. (2008), the results from the random effects probit model cannot be directly compared with the results from the pooled probit model because they use different normalizations. Rescaling by the suggested correction factor ($\sigma_u/\sigma_v=\sqrt{(1-\rho)}$), however, does not change the qualitative conclusions. For example, the rescaled estimate of γ for Model 3 in the comparison area was 0.1295.

In both areas, mother's schooling significantly reduces the risk of infant mortality for the first child, but is insignificant for higher birth orders once the father's schooling is also controlled for (Model 3). On the other hand, schooling of the father significantly reduces infant mortality of higher birth orders but not of first born children. It seems hard to interpret these differences; both schooling variables are measures of the family's socio-economic status, and the general conclusion is that higher socio-economic status implies lower mortality risks. The third indicator of (low) socio-economic status is a dummy indicating whether the father is a day labourer. It has the expected significantly positive effect for higher birth orders, but is insignificant for mortality of first born children.

Those who used tube well or pipe water as a source of drinking water are less likely to see their children die in infancy die but this finding is significant for higher birth orders in the ICDDR,B area only. The distance to the nearest health facility has a significantly positive effect on infant mortality in the comparison area, and the effect is particularly pronounced for the first born child. That no significant effect is found in the ICDDR,B area may be due to the fact that almost all families live rather close to a health facility in that area.

7. Decomposing the Difference between Areas

The aggregate prediction of the infant mortality rate according to model 3 for all children (first born as well as others) is about 49 per thousand live births in the ICDDR,B area and 67 per thousand live births in the comparison area, a difference of 18 per thousand live births. We analyze the gap between the two areas using the common technique of decomposing differences in mean levels into those due to different observable characteristics or "endowments" and those due to different effects of characteristics or "coefficients" (Blinder, 1973; Oaxaca, 1973). In the standard case of a linear model the technique requires coefficient estimates from linear regressions for the outcome of interest and sample means of the independent variables used in the regressions. Adjustments for the case of a nonlinear model such as our binary choice model were introduced by Fairlie (2005) and Yun (2004).

Here we follow the decomposition methodology proposed by Yun (2004) for the probit model (binary dependent variable), which can straightforwardly be extended to the dynamics random effects probit model taking into account of the initials conditions and the unobserved heterogeneity. The 'aggregate' or 'overall' mean difference in infant mortality

between the two areas ICDDR,B (group A) and comparison (group B) can be decomposed as follows:

$$\begin{aligned}\bar{Y}_A - \bar{Y}_B = &\left[\overline{\Phi(X_A\beta_A + \lambda_A\alpha_A)} - \overline{\Phi(X_B\beta_A + \lambda_A\alpha_B)}\right] + \\ &+\left[\overline{\Phi(X_B\beta_A + \lambda_A\alpha_B)} - \overline{\Phi(X_B\beta_B + \lambda_B\alpha_B)}\right]\end{aligned} \tag{8}$$

The means are taken over either all first born children or all higher birth orders of all mothers in each area and over the random effects. We have used shorthand notation, dropping indexes i and t and combining expressions for the first born child and the second and higher birth orders. For example, X_A includes x_{it} as well as y_{it-1} for *t>1,* β_A denotes either π (for birth order 1) or (β, γ) (for higher birth orders), and $\lambda = \theta$ or $\lambda = 1$ for birth order equal to 1 or larger than 1, respectively.

The first component in the decomposition in equation (8) is the "endowment" or "composition effect", the part of the difference explained by differences in (observed and unobserved) characteristics of in the two samples. The second is the residual difference keeping characteristics constant. To estimate the two components, we replace the parameters by the estimates for Model 3 in Table 3 for the treatment area (*A*) or the comparison area (*B*). This is referred to as decomposition 1. We also present the results in the reverse order, i.e., taking the comparison area as group A and the treatment area as group B (and adding minus signs for comparability), which we refer to as decomposition 2. The unobserved heterogeneity terms are replaced by random draws from their estimated normal distributions.

To understand which characteristics contribute to explaining the mortality difference between the two regions, we also performed the so-called detailed decomposition, again following Yun (2004). For this purpose, equation (8) is rewritten as follows:

$$\begin{aligned}\bar{Y}_A - \bar{Y}_B = &\sum_{i=1}^{i=k} W_{\Delta x}^i \left[\overline{\Phi(X_A\beta_A + \lambda_A\alpha_A)} - \overline{\Phi(X_B\beta_A + \lambda_A\alpha_B)}\right] \\ &+\sum_{i=1}^{i=k} W_{\Delta\beta}^i \left[\overline{\Phi(X_B\beta_A + \lambda_A\alpha_B)} - \overline{\Phi(X_B\beta_B + \lambda_B\alpha_B)}\right]\end{aligned} \tag{9}$$

where the "weights" are given by

$$W^i_{\Delta X} = \frac{\left(\overline{X}^i_A - \overline{X}^i_B\right)\beta^i_A}{\left(\overline{X}_A - \overline{X}_B\right)\beta_A} \text{ and } W^i_{\Delta\beta} = \frac{\left(\beta^i_A - \beta^i_B\right)\overline{X^i_B}}{\left(\beta_A - \beta_B\right)\overline{X_B}}, \text{ so that } \sum_{i=1}^{i=k} W^i_{\Delta x} = \sum_{i=1}^{i=k} W^i_{\Delta\beta} = 1.$$

We focus on the contribution of each variable to the endowment effect, the first part in (9). We present the results for the second part for completeness, but we do not have a good interpretation for these in our context.

The bottom rows of the two panels in Table 4 give the results of the overall decomposition ("Total"). For the first born child, almost two thirds of the mortality gap is explained by characteristics according to both decomposition 1 and decomposition 2 (16.3 or 17.0 per one thousand live births, of a total gap of 24.9 per thousand live births). The detailed composition shows that this is almost completely due to the variable *distance to the nearest health facility*. This variable has a strong (negative) effect on survival chances and the distances are much larger in the comparison area than in the treatment area.

For higher birth orders, differences in characteristics explain a smaller part of the total gap and the results are sensitive to which of the two decompositions is used. According to decomposition 2, the endowment effect is about one third of the total effect (5 out of the total gap of 15 per thousand live births) and again this is mainly driven by the distance to the nearest health facility, though mother's age at birth also plays a role: mortality falls with age of the mother at birth, and mothers in the treatment area are somewhat older, on average. According to decomposition 1, however, the difference in distance to a health facility hardly plays a role. The reason is that this is now weighted by the coefficient estimate for the distance variable in the treatment area, which is small and insignificant. Accordingly, decomposition 1 also attributes a much smaller contribution to all observed differences in characteristics (1.5 out of 15).

8. Discussion

We have analyzed the determinants of infant mortality in Bangladesh, in an area with and an area without health services beyond the standard services provided by the government. We have used recently developed methods accounting for heterogeneity across families as well as the within family dynamics of infant mortality – accounting for the two potential explanations for the stylized fact that a child has a larger mortality probability if the

previous child of the same mother died. Separating the causal effect from unobserved heterogeneity has important implications for policy in this area and for research on the inter-relations of family behaviour and mortality. Indeed, the causal effect of infant death of the previous child appears to be overestimated in a model without unobserved heterogeneity compared to the full dynamic model, showing the importance of controlling for mother specific unobserved heterogeneity.

We find a substantial and significant scarring effect in the comparison area. The likelihood of infant death is about 30% more if the older sibling died in infancy and the estimates suggest that, in the absence of scarring, the infant mortality rate among the second and higher order births would fall by 6.2%. Thus, policies targeted at reducing childhood mortality are important to also avoid the death of subsequent siblings. There is no evidence of scarring in the treatment area; a possible explanation of this is a (negative) learning effect that plays a role with available extensive health interventions and is large enough to annihilate the (positive) scarring effect. Mothers of the ICDDR,B area are routinely visited by the community health workers helping them to be resourceful with knowledge and health information. Another explanation might be that the mechanism through fertility and birth intervals (the death of a child leads to a shorter birth interval and short birth intervals lead to more vulnerable children) plays less of a role, because the better health services and information provisions limit the shortening of birth intervals after child death (the replacement effect). Further research identifying the latter mechanism (as in Bhalotra and van Soest, 2008) can disentangle these explanations. In any case, our result in this respect is in line with the finding by Sastry (1997) that clustering of mortality risks is greater in settings with high fertility and high mortality. Arulampalam and Bhalotra (2008) also found a weak scarring effect in more developed Indian states in India like Punjab (the richest state), and Kerala (socially the most advanced).

The aggregate level mother-specific variation in infant deaths is 18 percent of the total unsystematic variation in the ICDDR,B area, compared to only 8 percent in the comparison area. This difference can be explained as follows: The ICDDR,B area is divided into four sub-regions, so-called Blocks. ICDDR,B interventions are phased out at different times in different Blocks. For example, measles vaccination to all children started in 1982 in two Blocks and in 1985 in the other two Blocks. Thus, children of different birth cohorts under study benefited differently from these interventions. Another explanation is that some

mothers who receive health information are better at exploiting this than others so that the additional health information increases the heterogeneity in health outcomes.

Estimating the model for the higher educated mothers only (results not reported) suggests that the mother specific variation in infant deaths is 18 percent higher among mothers with secondary school or higher education than for the complete ICDDR,B sample. This finding confirms the statement that "the new interventions will tend to increase the inequality since they will initially reach those who are already better off" (Victora et al., 2000; Razzaque et al., 2007). On the other hand, in the situation without interventions in the comparison area, the unobserved heterogeneity is higher among the mothers with low education level, which may be due to variation in innate ability in this group (Das Gupta, 1990).

Comparison of other covariate effects between areas offers some interesting new insights. For children of birth order two and higher, the likelihood of infant death falls with the schooling years of father. In the comparison area, the mother's education plays no significant role keeping the father's education constant for the second and further children, but it does play a significant role in reducing the infant's death for the first-born child. The fact that occupation of the father matters - a day labourer is more likely to experience infant deaths - is similar to the finding of D'Souza and Bhuiya (1982). It might reflect the association between high mortality and poor socioeconomic conditions with insecure household income. We find that first children are more likely to die if they are boys than if they are girls in the ICDDR,B area, while a much smaller and in significant difference by gender is observed in the comparison area. The stylized fact seems to be that biologically a male child has a higher mortality probability during childhood than a female child (Majumder et al., 1997; Baragi et al., 1999), so that the insignificant difference in the comparison area may reflect some behavioural effect related to son preference.

A larger distance to the nearest health facility leads to higher mortality in the comparison area and this effect is more pronounced for the first born child than for children of higher birth order, possibly reflecting the social taboos of restricting the mobility of younger mothers. In line with this, decomposing the infant mortality gap between the two areas shows that *distance to the nearest health facility* plays an important role in explaining the between areas mortality gap for first born children in particular. This single factor '*distance to the nearest health facility*' explains 15 per 1,000 live births of the total difference of 25 per 1,000 live births among first-borns in the two areas. For higher birth

orders, the importance of the distance to the health clinic in explaining the infant mortality gap depends on which decomposition is used. In any case, it seems that the variable plays a larger role for first born children than for higher birth orders. Accordingly, the total set of observed characteristics explains a larger part of the gap for first-born children than for later births. This may be related to the fact that new mothers do not tend to visit health clinics outside the village due to social taboos.

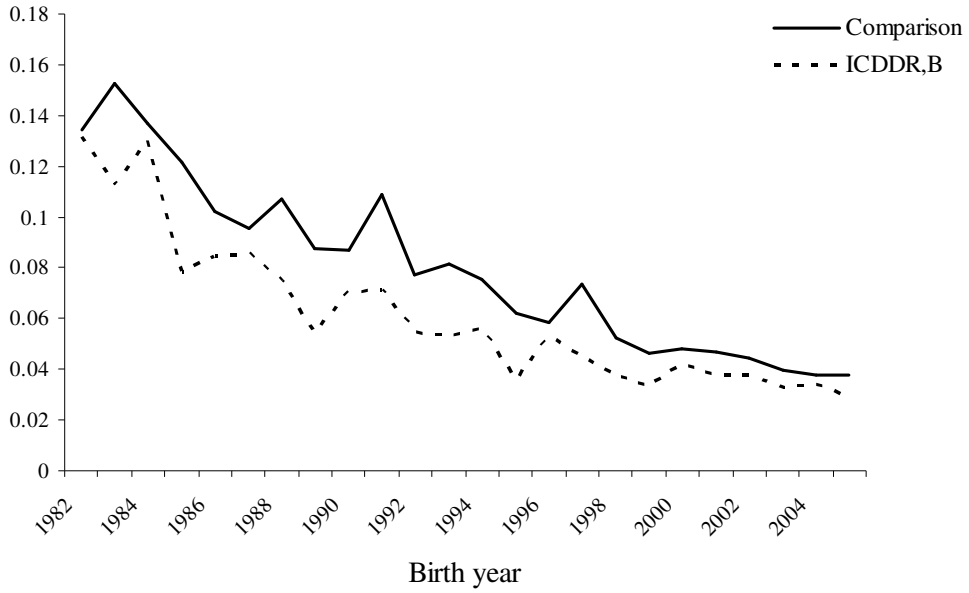

Fig. 1. Infant mortality rates by birth year

Table 1. Descriptive Statistics

Variables	Area	
	ICDDR,B	**Comparison**
% of infant deaths (all live-births)	5.00	6.74
% of infant deaths excluding first-borns	3.85	5.55
% of infant deaths among first borns	6.63	8.78
% families with no infant deaths	89.50	84.53
% families in which all births die in infancy	0.0091	0.0130
Age of mother at first birth*	21.16 (3.23)	21.08 (3.21)
Age of mother at birth*	24.70 (5.03)	24.58 (4.89)
Mother's years of schooling*	3.52 (3.75)	3.20 (3.56)
% mothers no schooling	42.47	50.50
% mothers 1-5 years of schooling	24.86	25.51
% mothers 6 or more years of schooling	26.66	24.0
% mothers Muslim	82.71	89.85
Number of children ever born per mother*	2.42 (1.30)	2.73 (1.53)
Number of children ever born per mother (%):		
1	30.10	27.01
2	27.34	23.14
3	23.18	20.62
4+	19.38	29.23
% first-born children	41.35	36.63
Father's years of schooling (father) *	2.93 (3.98)	2.68 (3.68)
% father no schooling	54.89	55.52
% father attended 1-5 years schooling	22.65	24.15
% father attended 6+ years schooling	21.68	19.57
% father day labourer	19.61	20.96
% families with drinking water source tubewell / piped water	87.12	76.91
Distance from nearest hospital (km) *	1.87 (0.98)	7.07(4.04)
Number of mothers in sample +	13,232	11,856
Number of children in sample ++	31,968	32,366

*: Means and standard deviations (in parentheses)
+ Sample mothers are who continue living in Matlab (never migrated out) since 1982 June to 2005 December after given first birth
++ All births in Matlab HDSS area from July 1982 to December 2005 for which survival status after twelve months is observed.

Table 2. Clustering and scarring in sibling infants deaths[1]

Estimates	Area	
	ICDDR,B	**Comparison**
(a) **Raw data**		
1 Incidence of infant death/1000 live births	50.0	67.4
2 Incidence of infant death/1000 live births excluding first-borns	38.5	55.5
3 Probability ($y_{ij}=1 \mid y_{ij-1}=1$), p_1	0.0796	0.1017
4 Probability ($y_{ij}=1 \mid y_{ij-1}=0$), p_0	0.0354	0.0510
5 Persistence due to y_{ij-1} (difference measure) (row 3- row 4), APR	0.0442	0.0507
6 Persistence due to y_{ij-1} (ratio measure) (row 3/row 4), PPR	2.2486	1.9941
(b) **Model estimates (Model 3)**		
7 Probability ($y_{ij}=1 \mid y_{ij-1}=1$), p_1	0.0315	0.0680
8 Probability ($y_{ij}=1 \mid y_{ij-1}=0$), p_0	0.0377	0.0526
9 Persistence due to y_{ij-1} (difference measure) (row 7- row8), APE	-0.0062	0.0154
10 Persistence due to y_{ij-1} (ratio measure) (row 7/row 8), PPR	0.8359	1.2933
11 % raw persistence explained (row 9/row 5)	-14.0271	30.3748
12 Predicted probability of infant death excluding first-borns	0.0386	0.0561
13 % reduction in mortality if $\gamma = 0$ (with respect of row 12) 1-(row8*/row 12)*100	-	6.2389
14 Variance of family level heterogeneity (standard error)	0.2221 (0.0417)	0.0855 (0.0280)
15 % variance explained by family level heterogeneity	18.1736	7.8766
Number of mothers in sample	13,232	11,856
Number of children in sample	31,968	32,366

Notes:

In rows 3 and 4 (part (a)), p_1 is the observed probability of infant death conditional on previous sibling died at infancy; p_0 is the observed probability of infants death conditional on previous sibling survived at infancy.

In rows 7 and 8 (panel (b)), p_1 is computed using the estimated marginal predicted probably of y_{it} for each observation under the condition previous sibling died at infancy ($y_{it-1} = 1$) and then averaging over all observations excluding the first borns. Similarly, p_0 is obtained as setting $y_{it-1} = 0$.

[1] This table is built up in a similar way as Table 2 in Arulampalam and Bhalotra (2006)

Table 3a. Estimation Results of Dynamic Random Effects Probit Models for Death at Infancy, Birth Order > 1

Covariates	ICDDR,B area			Comparison area		
	Model 1	**Model 2**	**Model 3**	**Model 1**	**Model 2**	**Model 3**
Previous sibling died (γ)	0.0646	-0.0869	-0.0918	0.1875	0.1270	0.1354
	(0.0770)	(0.0856)	(0.0858)	(0.0577)	(0.0621)	(0.0621)
Male		0.0488	0.0453		0.0142	0.0137
		(0.0384)	(0.0384)		(0.0304)	(0.0303)
Birth order		0.1126	0.1185		-0.1269	-0.1208
		(0.1018)	(0.1017)		(0.0542)	(0.0541)
Birth order square		-0.0186	-0.0185		0.0165	0.0162
		(0.0138)	(0.0137)		(0.0064)	(0.0064)
Mother's age at birth		-0.2103	-0.1980		-0.0689	-0.0638
		(0.0406)	(0.0408)		(0.0336)	(0.0337)
Mother's age at birth square		0.0034	0.0033		0.0008	0.0007
		(0.0007)	(0.0007)		(0.0006)	(0.0006)
Muslim		-0.0720	-0.0331		-0.0908	-0.0702
		(0.0529)	(0.0552)		(0.0493)	(0.0510)
Schooling years mother 1-5 years		-0.0953	-0.0593		-0.0298	0.0089
		(0.0504)	(0.0520)		(0.0390)	(0.0403)
Schooling years mother 6+ years		-0.1598	-0.0415		-0.1615	-0.0811
		(0.0614)	(0.0672)		(0.0517)	(0.0554)
Mother's birth cohort:						
1966-1970		-0.0219	-0.0008		-0.1676	-0.1583
		(0.0529)	(0.0533)		(0.0402)	(0.0408)
1971-1975		-0.1604	-0.1362		-0.3062	-0.3065
		(0.0623)	(0.0634)		(0.0473)	(0.0491)
1976+		-0.1885	-0.1559		-0.5575	-0.5512
		(0.0719)	(0.0736)		(0.0600)	(0.0625)
Schooling years father 1-5 years			0.0642			-0.0496
			(0.0494)			(0.0390)
Schooling years father 6+ years			-0.1861			-0.1455
			(0.0651)			(0.0494)
Father's occupation is day labourer			0.1331			0.0843
			(0.0520)			(0.0394)
Source of drinking water: tubewell / piped water			-0.1775			-0.0164
			(0.0604)			(0.0399)
Distance to health facility (km)			-0.0003			0.0086
			(0.0212)			(0.0039)
Constant	-1.9807	1.0723	0.8990	-1.7209	0.0540	-0.1197
	(0.0467)	(0.5389)	(0.5471)	(0.0278)	(0.4494)	(0.4561)

Notes:

Standard errors are in parentheses

Reference categories of categorical variables used in the model: female, non-Muslim, no schooling years, no access to piped water, not day labourer, mother born before 1966.

Table 3b. Estimation Results of Dynamic Random Effects Probit Models for Death at Infancy, First Born Children

Covariates	ICDDR,B area			Comparison area		
	Model 1	**Model 2**	**Model 3**	**Model 1**	**Model 2**	**Model 3**
Male		0.1217	0.1218		0.0659	0.0629
		(0.0372)	(0.0372)		(0.0342)	(0.0342)
Mother's age at birth		-0.1045	-0.1026		-0.1553	-0.1538
		(0.0383)	(0.0382)		(0.0358)	(0.0356)
Mother's age at birth square		0.0017	0.0017		0.0028	0.0029
		(0.0008)	(0.0008)		(0.0008)	(0.0007)
Muslim		-0.0165	-0.0018		-0.0387	-0.0042
		(0.0478)	(0.0497)		(0.0556)	(0.0571)
Schooling years mother		-0.2018	-0.1933		-0.1562	-0.1339
1-5 years		(0.0480)	(0.0497)		(0.0442)	(0.0456)
Schooling years mother		-0.3423	-0.2965		-0.3367	-0.3063
6+ years		(0.0511)	(0.0563)		(0.0484)	(0.0527)
Mother's birth cohort:						
1966-1970		-0.1451	-0.1409		0.0094	0.0228
		(0.0572)	(0.0575)		(0.0566)	(0.0571)
1971-1975		-0.1854	-0.1783		0.0048	0.0391
		(0.0595)	(0.0610)		(0.0588)	(0.0615)
1976+		-0.4200	-0.4123		-0.1783	-0.1245
		(0.0610)	(0.0632)		(0.0591)	(0.0640)
Schooling years father			0.0657			-0.0339
1-5 years			(0.0462)			(0.0428)
Schooling years father			-0.0744			-0.0049
6+ years			(0.0535)			(0.0487)
Father's occupation is			0.0285			0.0829
Day labourer			(0.0450)			(0.0420)
Source of drinking water:			-0.0353			-0.0582
tubewell / piped water			(0.0520)			(0.0425)
Distance to health facility			0.0183			0.0154
(km)			(0.0182)			(0.0042)
Constant	-1.6433	0.1321	0.0544	-1.4011	0.7885	0.5859
	(0.0550)	(0.4548)	(0.4605)	(0.0321)	(0.4242)	(0.4276)
ρ	0.1684	0.2283	0.2221	0.0977	0.0940	0.0855
	(0.0369)	(0.0415)	(0.0417)	(0.0241)	(0.0280)	(0.0280)
θ (see eq. 4)	0.9779	0.7436	0.7476	0.8054	0.7875	0.7803
	(0.2576)	(0.1693)	(0.1747)	(0.2748)	(0.3024)	(0.3296)
Log-likelihood	-6238	-6094	-6076	-7879	-7704	-7685

Note:

ρ is defined as $\frac{\sigma_\alpha^2}{\sigma_\alpha^2 + \sigma_u^2}$; it is the proportion of the total unsystematic variance that can be attributed to family-level unobservables α_i.

Table 3c. Average Predicted Probabilities Given Previous Sibling's Survival Status; Models 1, 2 and 3

	ICDDR,B area			Comparison area		
Probabilities	**Model 1**	**Model 2**	**Model3**	**Model 1**	**Model 2**	**Model 3**
p_1^*	0.0403	0.0372	0.0315	0.0726	0.0676	0.0680
p_0^*	0.0354	0.0417	0.0377	0.0511	0.0532	0.0526
APE: $p_{1-} p_0$	0.0049	-0.0045	-0.0062	0.0216	0.0144	0.0154
PPR: p_1/p_0	1.136	0.8921	0.8359	1.422	1.2711	1.2933

Note: Probabilities are computed in the same way as $p_{1,}$ p_0 in panel b of Table 2

Table 4: Decomposition of Differences in Infant Mortality Rates between ICDDR,B and Comparison Area

	Decomposition 1			
	Child=1		Child>=2	
Variables used in the model	Diff. in Characteristics	Diff. in Coefficients	Diff. in Characteristics	Diff. in Coefficients
Distance to health facility	-14.825	-0.443	0.098	-0.63366
Mother's schooling years	-0.360	0.264	-0.046	-0.10362
Mother's age at birth	-0.309	-12.048	-0.679	-17.195
Mother's birth cohort	0.417	4.601	0.066	1.673
Father's schooling years	-0.486	-0.190	-0.226	0.201
Father's occupation	-0.058	0.301	-0.161	0.091
Birth order	-	-	0.312	3.583
Male	-0.042	-0.666	-0.004	0.161
Muslim	0.023	-0.046	0.128	0.333
Previous child died	-	-	0.107	-0.204
Source of drinking water	-0.645	-0.376	-1.186	-1.264
Total	-16.284	-8.603	-1.591	-13.359

	Decomposition 2			
	Child=1		Child>=2	
Variables used in the model	Diff. in Characteristics	Diff. in Coefficients	Diff. in Characteristics	Diff. in Coefficients
Distance to health hospital	-14.877	-0.111	-3.924	-0.127
Mother's schooling years	-0.656	0.224	-0.122	-0.074
Mother's age at birth	-0.348	-1.137	-1.184	-13.074
Mother's birth cohort	-0.026	4.307	0.511	1.237
Father's schooling years	0.108	-0.103	-0.143	0.138
Father's occupation	-0.199	0.269	-0.141	0.062
Birth order	-	-	-0.023	0.277
Male	-0.026	-0.626	-0.002	0.122
Muslim	0.061	-0.040	0.376	0.237
Previous child died	-	-	-0.219	-0.123
Source of drinking water	-1.267	-0.410	-0.152	-1.0934
Total	-17.030	-7.860	-5.023	-9.928

A=ICDDR,B area; B=Comparison area

Annex

Table A1. Estimation Results of Pooled Dynamic Probit Models for Death at Infancy, Birth Order >1

	ICDDR,B area			Comparison area		
Covariates	**Model 1**	**Model 2**	**Model 3**	**Model 1**	**Model 2**	**Model 3**
Previous sibling died (γ)			0.2996			0.2828
			(0.0558)			(0.0445)
Male			0.0372			0.0138
			(0.0342)			(0.0291)
Birth order			0.1311			-0.1180
			(0.0912)			(0.0512)
Birth order square			-0.0123			0.0185
			(0.0122)			(0.0060)
Mother's age at birth			-0.1590			-0.0539
			(0.0353)			(0.0319)
Mother's age at birth square			0.0026			0.0006
			(0.0006)			(0.0006)
Muslim			-0.0317			-0.0650
			(0.0470)			(0.0474)
Schooling years mother			-0.0447			0.0148
1-5 years			(0.0440)			(0.0375)
Schooling years mother			-0.0026			-0.0624
6+ years			(0.0570)			(0.0519)
Mother's birth cohort:						
1966-1970			-0.0012			-0.1525
			(0.0445)			(0.03762)
1971-1975			-0.1071			-0.2881
			(0.05357)			(0.0455)
1976+			-0.1156			-0.5169
			(0.0628)			(0.0580)
Schooling years father			0.0617			-0.0524
1-5 years			(0.0418)			(0.0364)
Schooling years father			-0.1548			-0.1408
6+ years			(0.5531)			(0.0463)
Father's occupation is			0.1239			0.0860
day labourer			(0.0451)			(0.0372)
Source of drinking water:			-0.1527			-0.0190
tubewell / piped water			(0.0522)			(0.0376)
Distance to health facility			-0.0002			0.0081
(km)			(0.0180)			(0.0037)
Constant			0.4903			-0.2260
			(0.4815)			(0.4336)
Log likelihood			-2986			-4266

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